IN THE UNITED STATES DISTRICT COURT FOR THE WESTERN DISTRICT OF TENNESSEE MEMPHIS DIVISION

JESSE LEE WHITE, **Jr.**, Deceased, by and through his Wife and Next Friend, **SHARON WHITE**, and **SHARON WHITE**, Individually Plaintiff vs. **UNITED STATES OF AMERICA**, Defendant	**NO.** _____

ORIGINAL COMPLAINT

Plaintiff **JESSE LEE WHITE**, **Jr.**, Deceased, by and through his Wife and Next Friend, **SHARON WHITE**, and **SHARON WHITE**, Individually, brings this complaint under the Federal Tort Claims Act, 28 U.S.C. § 2674. Plaintiff complains of the United States and would show the following.

PARTIES

1.1. This case arises out of bodily injuries caused by agents and employees of the United States at the Memphis VA Medical Center in Memphis, Tennessee.

1.2. Plaintiff **Sharon White** is the wife and next friend of Plaintiff-decedent, **JESSE LEE WHITE**, **Jr.** She brings this action individually and as the personal representative of Jesse Lee White, Jr., and is the proper person to do so in her

representative capacity. At all pertinent times, Plaintiffs were residents of Shelby County, Tennessee.

1.3. Defendant is the United States of America.

JURISDICTION, SERVICE & VENUE

2.1. This Federal District Court has jurisdiction because this action is brought under 28 U.S.C. § 2671–80, commonly known as the Federal Tort Claims Act.

2.2. The United States of America may be served with process in accordance with Rule 4(i) of the Federal Rules of Civil Procedure by serving a copy of the Summons and Complaint on the United States Attorney D. Michael Dunavant, United States Attorney for the Western District of Tennessee, by certified mail, return receipt requested at his office:

> The United States Attorney's Office
> ATTN: Civil Process Clerk
> Clifford Davis/Odell Horton Federal Building
> 167 N. Main Street #800
> Memphis, TN 38103

2.3. Service is also affected by serving a copy of the Summons and Complaint on William Barr, Attorney General of the United States, by certified mail, return receipt requested at:

The Attorney General's Office
ATTN: Civil Process Clerk
950 Pennsylvania Avenue, NW
Washington, DC 20530-0001

2.4. Venue is proper in this judicial district under 28 U.S.C. § 1402(b) because the United States of America is a defendant and the acts and omissions complained of in this lawsuit occurred in this judicial district.

AGENCY

3.1. This case is commenced and prosecuted against the United States of America to and in compliance with Title 28 U.S.C. §§ 2671–80, the Federal Tort Claims Act. Liability of the United States is predicated specifically on 28 U.S.C. § 2674 because the personal injuries and resulting damages of which the complaint is made were proximately caused by the negligence, wrongful acts and/or omissions of employees and/or agents of the United States of America working for the Veterans Affairs, while acting within the scope of their office, employment, and/or agency under circumstances where the United States of America, if a private person, would be liable to the Plaintiff in the same manner and to the same extent as a private individual.

3.2. The United States Department of Veterans Affairs (VA) is an agency of the United States of America.

3.3. The United States of America, through its agency, the VA, at all times material to this lawsuit, owned, operated, and controlled the Memphis VA Medical Center, in Memphis, TN, and staffed it with its agents, servants, or employees.

3.4. At all times material to this lawsuit, all medical providers involved in the care and treatment of Jesse Lee White, Jr. were acting within the course and scope of their employment with the Memphis VA Medical Center when providing treatment to Mr. White.

3.5. The Memphis VA provided care and treatment to Mr. Jesse Lee White, Jr. Mr. White was a patient of the Memphis VA and the Memphis VA and its providers had a doctor-patient relationship with Mr. White.

JURISDICTIONAL PREREQUISITES

4.1. Pursuant to 28 U.S.C. §§ 2672 and 2675(a), the claims set forth here were filed with and presented administratively to the Department of Veterans Affairs on March 27, 2019 and received by the Department of Veterans Affairs on April 1, 2019. The VA denied via certified mail the administrative claim by letter dated January 22, 2020. Request for Reconsideration was filed by Plaintiff on April 30, 2020. The VA has yet to finally deny the claim or act on the Request for Reconsideration, and more than six months has elapsed since Request for Reconsideration was filed.

4.2. Accordingly, Plaintiff has complied with all jurisdictional prerequisites and conditions precedent to the commencement and prosecution of this suit.

FACTS

5.1. On October 19, 2017, Mr. White, age 59 at the time of admission, presented to the Memphis VA Medical Center, reporting muscle weakness, back pain, dark urine and immobility. The initial Internal Medicine note recounted a history of Mr. White becoming bed bound for about a week prior to admission due to his legs suddenly becoming very weak, as well as back pain increasing in intensity and urine becoming dark over the last day - all signs of rhabdomyolysis, confirmed by a CPK (Creatinine Phosphokinase) of 11,117. A CT and MRI of his lumbar spine were done; the MRI was found to be unchanged from a prior study in 8/2016. He was diagnosed with rhabdomyolysis.

5.2. The attending Nephrologist evaluated Mr. White on the 20th of October, and included in his history a recent fall due to his knee giving out on him with his urine becoming darker than usual at about that time. Acute kidney injury was noted secondary to the rhabdomyolysis, and IV fluid hydration was agreed to be indicated. However, examination revealed IV fluid hydration was inadequate, as his skin turgor was poor and his blood pressures continued to be low.

5.3. The Internal Medicine note later that afternoon was significant in that Mr. White was still complaining of weakness and also of thigh pain, and emphasized

that his weakness was more acute in onset. Exam revealed his right thigh was slightly edematous as compared to the left.

5.4. Evaluation by the neurologist, also on October 20th, indicated that about a week previous, Mr. White developed worsening neck and lower back pain and new weakness which began with episodic knee buckling while ambulating and inability to rise from a seated position. That inability resulted in his being confined to bed and losing "50 pounds" while bedbound. Probable Statin Myopathy was diagnosed due to the sudden onset of weakness, especially since it was not associated with a sensory deficit. This neurologist noted that there were two forms of statin myopathy: the self -limited non- immune form and the autoimmune form. Regardless of type, he expected the myopathy to respond to discontinuation of the statin before 21 days and that Mr. White would return to baseline within 2 to 14 months. As the statin myopathies related to autoimmune disease show significant necrosis without significant inflammation, he proposed a full workup and muscle biopsy be performed if the symptoms did not improve. He suggested that these studies would be necessary prior to long-term and/or aggressive immunotherapy. He further noted that Anti-HMGCoAR antibodies were available at Dr. Pestronk's St. Louis University laboratory at no charge.

5.5. As of October 20th, testing of the nares for the presence of methicillin resistant staphylococcus aureus (MRSA) was negative. On the 21st, the Neurology Fellow, Dr. Kuwabara, wrote that immunosuppressive therapy was not indicated at that time, as Mr. White was improving. Dr. Kuwabara further noted that he would consider sending anti-HMGCoAR antibodies if clinical improvement was seen in 3 weeks.

5.6. The CPK isoenzyme was noted to be 8820 at that time (down from 10,355) with the Creatinine and BUN levels (reflecting kidney function) decreased to 3.5 and 47 respectively (down from 3.9 and 49). Mr. White stated that his strength was improving. However, he also complained of pain when his thighs were squeezed. On this date, a urinalysis was performed that revealed 3+ blood, a pH of 5 (acidotic), and a specific gravity of 1.011 (specific gravity above 1.010 is indicative of mild dehydration). These results should have informed the health care providers that insufficient hydration was ongoing and that the administration of sodium bicarbonate had not been sufficient to keep the urine pH > 6.5.

5.7. By October 24, Mr. White's CK and AST were rising. Steroids were ordered for his increased CPK levels. Rheumatology had been consulted and an antibody panel was drawn pursuant to their recommendation.

5.8. Examination the afternoon of the 24th included notes that Mr. White was now reporting pain "everywhere," but especially in his feet after a wound care procedure that day. Mild edema was noted on exam in both lower extremities. Assessment was noted to be rhabdomyolysis secondary to immobility, statins, and obesity with autoimmune etiology on the differential diagnosis per neurology. The plan was still to consider immunosuppressive therapy only if he failed to improve or continued to worsen. The resident who evaluated Mr. White the morning of the 24th noted that a statin-associated myopathy was suspected over an autoimmune myopathy. This note was co-signed by the Assistant Chief of Neurology, Dr. Michael Jacewicz. In an addendum, a neurology resident, Dr. Mays, noted that Mr. White was improving clinically, and that the case would be discussed with their neuromuscular specialist.

5.9. By that afternoon, staff physician Dr. Postlethwaite had assessed Mr. White. He noted that Mr. White likely had necrotizing myopathy secondary to atorvastatin 40mg/day for several years, and that on admission, he had proximal muscle weakness of his upper and lower extremities and myoglobinuria (however, there no mention of upper extremity weakness on admission). He further noted that Mr. White was unable to lift his legs from the bed against gravity or sit up. Dr. Postlethwaite agreed with the plan to obtain autoantibody panels and then recommended he be treated with solumedrol 40mg BID (twice/day) and try CellCept as a steroid sparing agent "eventually." Finally, he added that it would be desirable to get a muscle biopsy to firmly establish the diagnosis.

5.10. As mentioned above, Mr. White's CPK levels had started rising by this time - from a low of 8820 on the 21st to 10,830 and 14,838 on the 22nd, 24,907 to > 32,000 on the 23rd (the laboratory was unable to discriminate values > 32,000) and it remained > 32,000 on the 24th and 25th. Internal Medicine notes on the 25th noted that Mr. White continued to have decreased intake by mouth and was complaining about pain in his left lateral foot that began two days prior (he had a dime sized bruise and point tenderness over the lateral 5th metatarsal). Sodium bicarbonate was restarted per the nephrology recommendations made on the 20th.

5.11. Despite the fact that the results of the various antibody tests had not returned, ongoing infection had not been ruled out, and a muscle biopsy had not been performed, aggressive immunosuppressive therapy was begun on October 26th with the administration of CellCept 250 mg BID (twice a day). Another urinalysis performed on the 26th revealed 3+ Leukocyte esterase, but no urine culture was

obtained. Such a culture would have provided information concerning an ongoing infection in the setting of a powerful immunosuppressant.

5.12. The surgery service was requested to review Mr. White's chart and lab results and agreed with plans of house staff, but believed that performing a muscle biopsy during active rhabdomyolysis would not be beneficial or diagnostic. Examination revealed that Mr. White could now raise his proximal legs against gravity but only slightly, that his abdominal muscles remained weak, and that he could not elevate his upper arms above the horizontal.

5.13. On October 31st, notes by Dr. Postlethwaite indicated that there was marked improvement of Mr. White's CPK down to 3,000. At that time, there was some movement of his distal upper and lower extremities, but none of the proximal muscle flexors of the thighs or elevation of the shoulders. Impression was of statin-induced myonecrosis, likely the anti-HMGCoA Reductase mediated type. The continued administration of CellCept was ordered. Dr. Postlethwaite also noted that biopsies were done on acute statin-induced myonecrosis patients in the medical literature.

5.14. By November 1st, Mr. White was found to be generally weak and fatigued, and had suffered from hypotension over the previous 24 to 48 hours. Monitoring for blood loss, infection and hypovolemia was considered. In addition, his serum bicarbonate levels were found to be low, and he had a lactate of 2.7.

5.15. MRSA testing was repeated and found to now be positive. That afternoon, a Rapid Response was called for a very low blood pressure of 66/42. Additional labs were drawn and an EKG was performed that revealed a prolonged QTc without ST changes. A liter of IV fluids was given as a bolus and the IV fluids were restarted at 100cc/hr.

5.16. About an hour after the Rapid Response had been called, Mr. White complained of not being able to breathe well; oxygen was placed and a portable chest x-ray and CT scan were done. After a total of 3 liters of IV fluid was infused, Mr. White's blood pressure only increased minimally, so the decision was made to move him to MICU.

5.17. Overnight a norepinephrine drip was started, vasopressin was added for additional pressor support and a mixed venous blood gas revealed acidosis with a pH of 7.27. Mr. White complained of worsening pain and redness of the left thigh, and the pulses in his lower extremities were found to be weak and thready. Surgery was then consulted for evaluation of possible compartment syndrome. Mr. White was also started on Clindamycin for possible infection.

5.18. By November 2nd, the diagnosis of septic shock secondary to left thigh cellulitis with possible necrotizing fasciitis and possible toxic shock syndrome had been made. CT performed of his chest revealed bibasilar atelectasis and small bilateral pleural effusions and CT of the abdomen/pelvis revealed moderate ascites and possible mesenteric adenopathy. Blood cultures were positive for gram negative rods and repeat urinalysis was consistent with a urinary tract infection. He was treated with broad spectrum antibiotics and multiple pressors for blood pressure support.

5.19. CellCept (mycophenalate mofetil) and methylprednisone were both discontinued on the 2nd after the septic shock was diagnosed. Previously drawn rheumatology labs all returned negative; they were still waiting for the results from the HMGCoA reductase antibody.

5.20. A diagnostic exploration of the left thigh was performed on November 3rd secondary to the gram-negative sepsis, septic shock, multi-organ failure and suspected left thigh necrotizing fasciitis. Left thigh cellulitis with compartment syndrome of the medial and anterior thigh compartments were found and released.

5.21. By November 4th, Mr. White was intubated and sedated with 4+ pitting edema of his upper and lower extremities, large incisions on his left thigh, and necrosis of the quadriceps. He was also tachycardic and became anuric with the need for vascular catheter placement for dialysis. As the HMGCoA reductase antibody value was 4 (range 1-19), it was finally decided that this was less likely a picture of statin induced myositis. The urine culture was returned and found to be positive for gram negative rods. He had worsening kidney function and anasarca (widespread swelling of the skin due to excessive fluid in the intravascular space).

5.22. On November 6th, the infectious disease consultant noted that Mr. White had initially been treated with steroids and CellCept due to thinking this was an autoimmune statin-induced myositis, but that he had developed leukopenia and septic shock with respiratory failure, requiring triple pressor support. He further noted that the autoimmune process had since been ruled out, since the HMGCoA Ab was negative. Mr. White was being treated for Klebsiella bacteremia and urinary infection (Klebsiella and Enterococcus), and critically ill.

5.23. Despite the use of maximum medical therapies with broad spectrum antibiotics, ventilator support, blood pressure support, and hemodialysis, Mr. White remained in profound vasodilatory shock and multi-organ failure with encephalopathy, respiratory failure, renal failure, and hepatic failure. Mrs. White

was finally convinced of his poor prognosis and agreed to a DNR (do not resuscitate). Mr. White died on November 17, 2018.

5.24. The autopsy performed on Mr. White revealed the cause of death to be multi-lobar consolidation (probable diffuse pulmonary hemorrhages and possible pneumonia) with disseminated intravascular coagulation and multi-system organ failure related to bacterial urinary tract infection complicated by bacteremia.

CAUSES OF ACTION

6.1. Through its employees, agents, or servants, the Defendant, United States of America, was negligent in one or more of the following respects:

(a) Negligently failing to timely order a urine culture;

(b) Negligently failing to rule out an infection prior to administering potent immunosuppressant therapy;

(c) Negligently administering potent immunosuppressant therapy prior to confirming that it was indicated;

(d) Negligently failing to obtain proper informed consent for the treatment rendered;

(e) Negligently failing to timely and properly care for Mr. White;

(f) Negligently failing to timely and properly evaluate Mr. White;

(g) Negligently failing to exercise that degree of skill and care as required of reasonable, prudent healthcare providers under the same or similar circumstances;

(h) Negligently and carelessly deviating from the standard of care that is required and expected of Defendants as they existed under the circumstances;

6.2. Had the health care providers adhered to the requisite standard of care, Jesse Lee White would have received the appropriate treatment and would not have suffered injuries, damages, and untimely death.

6.3. At all times relevant to this lawsuit, the officers, employees, agents, or representatives of the United States were negligent and caused the injuries and damages sustained by the Plaintiff.

DAMAGES

7.1. Because of the negligence of the United States employee healthcare providers, Jesse Lee White, Jr. suffered severe injuries, including:

a. Great fright and shock;
b. Great physical pain and suffering;
c. Great mental and emotional anguish;
d. Large medical expenses;
e. Untimely and wrongful death;

f. Funeral expenses;

g. Loss of the enjoyment of life; and

h. All other damages recoverable under the laws of Tennessee.

7.2. As a result of the negligence of the United States employee healthcare providers, Sharon White suffered loss of consortium and severe and extreme emotional distress in her role as wife of Jesse Lee White, Jr.

COMPLIANCE WITH TN ST § 29-26-121

8.1. While Tennessee Statute § 29-26-121 is a procedural statute inapplicable in a FTCA lawsuit in federal court, out of an abundance of caution, Plaintiff is filing a certificate of good faith contemporaneously with this Complaint.

RELIEF REQUESTED

Plaintiff requests that the United States be cited in terms of law to appear and answer this lawsuit. Upon final trial, Plaintiff seeks judgment against the United States for the amount of actual damages and for such other and different amounts that she shall show by proper amendment before trial; for post-judgment interest at the applicable legal rate; for all Court costs incurred in the prosecution of this lawsuit; and for such other relief, in law or equity, both general and special, to which the Plaintiff may show herself entitled to and to which the Court believes her deserving.

Respectfully Submitted,

s/ Timothy Holton
TIMOTHY HOLTON
TN State Bar #11832
tholton@holtonlaw.com
HOLTON LAW FIRM, PLLC
296 Washington Avenue
Memphis, TN 38103
(901) 523-2222 (o)

s/ Michelle Cheng
MICHELLE CHENG, *pro hac vice application forthcoming*
Texas State Bar No. 00796345
mcheng@nationaltriallaw.com
TOM JACOB, *pro hac vice application forthcoming*
Texas State Bar #24069981
tjacob@nationaltriallaw.com
WHITEHURST, HARKNESS, BREES, CHENG, ALSAFFAR, HIGGINBOTHAM, & JACOB P.L.L.C.
7500 Rialto Blvd, Bldg. Two, Ste 250
Austin, TX 78735
(512) 476-4346 (o)
(512) 467-4400 (f)

Attorneys for the Plaintiff